Case 2

Anne D. was an Indian girl of 16 years, the wife of the first patient described. The history and physical findings were similar to those of her husband. She was approximately five months pregnant and had apparently received no antepartum care. Urinalysis showed many leukocytes and culture of a mid-stream urine specimen produced a heavy growth of Enterococcus. Her ESR was 40 mm. and chest x-ray was normal. A biopsy of the gastrocnemius muscle confirmed the presence of larvae of *Trichinella spiralis*.

She was started on prednisone, 40 mg. per day, on the sixth hospital day, and discharged on a dosage of 7.5 mg. four times daily.

On March 19, 1971 she delivered a full-term normal male infant weighing 8½ lbs., which appeared perfectly healthy on careful examination. A year after her original infestation the mother appeared in good health, but a persistent leukocytosis of 11,000 per c.mm. was present, with 7% eosinophils. The cause of this is not known; no investigations for parasites were done at this time.

Discussion

The last reported outbreaks of trichinosis in Canada were due to pork.[1, 2] In one outbreak thiabendazole was used in the treatment of 11 cases but the author concluded that it conferred no definite benefit.[1]

The black bear has been recognized as a vector of trichinosis in the Arctic and cases in Alaska, Labrador and Northern Russia are reported.[3-5] We are not aware of a previous report from the Prairie provinces.

The transplacental transmission of trichinosis with intrauterine infestation of the fetus has been reported in experimental animals, and in presumptive cases in humans.[6, 7] The paucity of reports suggests that this must be a rare occurrence; despite the presumably heavy infection in Case 2, no abnormality was found in the child at delivery.

Résumé

Epidémie de trichinose en Saskatchewan, causée par consommation de viande d'ours avariée

Les auteurs rapportent une épidémie de trichinose dans une tribu d'Indiens Chippewayens vivant dans le Nord de la Saskatchewan et qui avaient mangé de la viande d'ours insuffisamment cuite. Le diagnostic a été basé sur l'anamnèse et sur l'examen clinique et confirmé par biopsie musculaire et par la découverte d'une dorte éosinophilie. Les sept malades ont guéri. Les deux malades qui ont été étudiés de façon plus approfondie ont été traités uniquement par la prednisone et ont guéri sans incidents: un an plus tard, on ne notait aucune séquelle. Une femme qui était enceinte de cinq mois au moment de l'infection a donné le jour à un enfant à terme et parfaitement normal.

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Trichinosis presenting as acute myocardial infarction

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Summary: A 20-year-old male patient is presented as a case of trichinous myocarditis with clinical symptoms and electrocardiographic evidence of an acute inferior myocardial infarction. He recovered rapidly and completely without any specific therapy. This seems to be a distinct rarity, having never been previously reported, but is of importance because of the almost uniformly excellent prognosis in this condition in contradistinction to that of a *bona fide* myocardial infarction occurring at this age.

Although in 1936 Cushing[1] stated that "cases of trichinosis with clinical and ECG evidence of myocardial damage are rare", more recent reviews have shown that among patients with clinically apparent trichinosis, myocardial involvement as witnessed by ECG abnormalities occurs in between 21 and 23%.[2, 3] These abnormalities include conduction defects, non-specific T-wave flattening or inversion, and arrhythmias.[1, 2, 4-6] However, there has never been reported a case of trichinous myocarditis presenting with focal ischemic changes resembling an acute myocardial infarction. Such a case was seen at the Royal Victoria Hospital in July 1968.

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Case report

A 20-year-old Sicilian-born male restaurant kitchen helper was admitted because of severe retrosternal pain of several hours' duration. Family history was entirely negative and the patient had never been ill prior to this episode. He had not left Canada since his arrival two years previously.

Two days before admission the patient noted low grade fever, malaise, and vague soreness in his shoulders and thighs. On the day of admission he experienced three episodes of severe dull retrosternal pain without radiation, each lasting 10 minutes, and all occurring within a few hours. He did not vomit, nor did he experience dyspnea, orthopnea, or other discomfort. He had not had a sore throat, joint pains, urinary discolouration or other symptoms.

Physical examination revealed a healthy looking young man in moderate distress due to chest pain as described above. Oral temperature was 100° F., pulse was 88 and regular, blood pressure was 120/80. He had 1+ periorbital edema but no edema elsewhere. Findings on examination of the chest were normal. Auscultation of the heart revealed an atrial gallop without other abnormalities. There was slight deltoid tenderness but no neurological or any other abnormality.

An electrocardiogram done in the emergency room showed insignificant

q waves in leads II, III, and avF, ST elevation in these leads and in V-6, and reciprocal ST depression in leads V-1-V-2 (Fig. 1). This was interpreted as probable acute inferior wall myocardial infarction and he was admitted to the cardiac monitor unit. Laboratory tests revealed a hemoglobin of 14.7 g., hematocrit 49%, and leukocyte count 12,300 with 6% eosinophilia. ESR was 4 mm. in the first hour. BUN, serum creatinine, electrolytes, uric acid, cholesterol, two hour p.c. blood sugar, PBI, serum electrophoresis, LE preparations, anti-nuclear antibodies, rheumatoid factor, antistreptolysin-o titer serum complement and Paul Bunnel test were all within normal limits. Cultures from the throat and of stool and urine for virus were negative, as were all routine bacterial cultures. SGOT was 46 units per 100 ml. on admission, and fell on the second day to 16, remaining normal thereafter; LDH was normal throughout. Chest x-ray was negative.

Serial ECGs revealed a very rapid sequence of changes. A second tracing done six hours after admission showed decreasing ST elevation in II, III, and avF, and new T wave inversion in lead III. Twelve hours later the T wave was inverted in II, III, and avF (Fig. 2). By the next day this inversion was less, and within three days the ST and T wave changes were minimal and found only in lead III (Fig. 3). Over the next few weeks ECGs continued to demonstrate variable changes indicative of inferior wall ischemia. There was never any conduction defect or arrhythmia.

The eosinophilia noted on admission increased to a maximum of 22% on the tenth hospital day and was 13% by the third week of hospitalization. Because of the absence of enzyme, leukocyte count and ESR changes, the unusual sequence of ECG changes, the eosinophilia, and the periorbital edema, a trichina skin test and trichinella agglutination test were done, both of which were weakly positive. The agglutination test repeated three weeks later was strongly positive, indicating a four-fold increase in the level of antibody. At this point the patient admitted to eating raw pork at least once weekly when the meat arrived at the restaurant in which he worked. X-rays of soft tissues were normal, as was an EEG. Muscle biopsy done two weeks after admission showed widespread myositis with eosinophilic and polymorphonuclear infiltration, but no larvae were detected.

The patient was treated by watchful waiting. His chest pain did not recur in hospital and fever, which varied between 101° and 102° during the first four days, disappeared thereafter. There was never evidence of cardiac failure, and the atrial gallop heard on admission soon disappeared leaving no abnormalities detectable by physical examination. When the diagnosis of trichinous myocarditis was considered firm, the patient was given a three-day course of thiabendazole which produced no change in his then asymptomatic course. When seen in clinic several months later, he had had no return of his symptoms and the ECG was normal. He had given up eating raw pork.

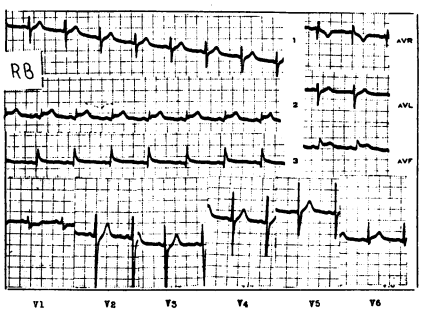

FIG. 1—Electrocardiogram taken at the time of admission to the hospital.

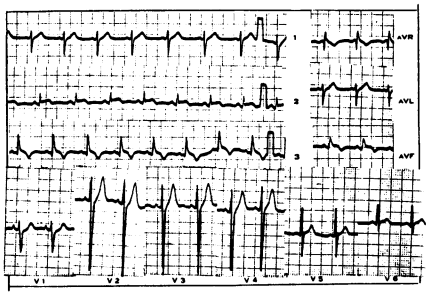

FIG. 2—Electrocardiogram taken 18 hours after admission.

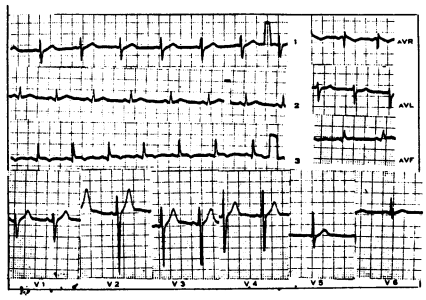

FIG. 3—Electrocardiogram taken on the fourth hospital day.

Comment

Despite the many cases of myocardial involvement in trichinosis reported in the literature, not one example could be found with ST segment elevation and the ECG signs of focal myocardial ischemia exhibited by our patient. That he had trichinosis and not another form of myocarditis seems sure. Although larvae were not found in the muscle biopsy, it should be remembered that biopsy performed earlier than the third week in the course of the disease may be unrewarding in this respect, and that even in the absence of demonstrable larvae, focal areas of acute necrosis with eosinophilic and polymorphonuclear infiltrations with or without associated angiitis are consistent with the diagnosis.[2, 7] The four-fold increase in antibody titre as shown by serum trichinella agglutination is of great diagnostic significance.[8] Of interest is the low ESR throughout this patient's course which has been reported in trichinosis.[9]

It is of paramount importance to distinguish this disease from a true myocardial infarction because of the great difference in prognosis between the two conditions. That a *bona fide* myocardial infarction occurring at age 20 is of grave prognostic importance is well known; the outcome of trichinous myocarditis is quite different. In reviewing 1023 patients, Gray found that although 23% of those with trichinosis had myocardial involvement, only one fatality could be attributed to the myocarditis.[2] That this serious complication of trichinosis is usually reversible is explained by its pathology. Zenker,[10] in 1860, was the first to demonstrate larvae in the myocardium of a patient who died of trichinosis myocarditis. The condition produces an active cellular proliferation, with localized areas of necrosis and hemorrhage scattered throughout the heart muscle.[11] These changes are thought to be due to the occurrence and migration of larvae in the myocardium. However, no encystment has ever been found in heart muscle, despite both clinical and experimental studies on the subject.[12, 13]

Because the larvae disappear from the myocardium, usually within two weeks, and no permanent encystment ever takes place therein, even the most serious cases of trichinous myocarditis will remit, providing proper treatment is given during the acute phase of the disease. Treatment, when there is evidence of serious cardiac compromise, consists of ACTH or corticosteroid therapy in high doses,[9, 14] along with careful monitoring of cardiac function and treatment of failure if need arises.

Because the treatment and prognosis of trichinous myocarditis differ from these aspects of acute myocardial infarction, we feel this relatively rare situation is worthy of serious consideration in any youth who presents with symptoms and ECG evidence of acute myocardial infarction, especially if any of the other stigmata of trichinosis are

present, such as periorbital edema, muscle aching or eosinophilia, or if there is a history of ingestion of raw pork.

Résumé

Cas de trichinose se présentant sous la forme d'un infarctus aigu du myocarde

Un homme de 20 ans se présente comme un cas de myocardite trichineuse, dont les symptômes cliniques et les signes de l'ECG évoquent un infarctus aigu du myocarde. Le malade a guéri rapidement et complètement sans recours à un traitement spécifique quelconque. D'après nous, ce cas est une entité clinique rare qui n'a jamais été rapportée. Il a cependant une réelle importance, étant donné l'excellent pronostic de cette pathologie, par opposition au cas classique de l'infarctus du myocarde qui survient à cet âge.

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